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Interleukin-33 in asthma: insights into pro-inflammatory roles of airway structural cells

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Background

Interleukin-33 (IL-33) is a novel cytokine that triggers inflammatory immune responses, but evidence of its role in human asthma, a common allergic airway disease, is lacking. There is also a paucity of information regarding which cells express IL-33, and what conditions promote its expression. We sought to investigate whether IL-33 is expressed in the lung tissue from patients with asthma.

Methods

We obtained lung biopsy tissue specimens from asthmatic adults and from healthy control subjects, along with normal primary cells from human airways that were cultured *in vitro*. We studied expression of IL-33 in lung tissue specimens, and determined whether conditions seen in asthma promote IL-33 expression *in vitro*. We also assessed whether IL-33 expression is sensitive to glucocorticoid treatment.

Results

Higher expression of IL-33 is detected in lung tissue from asthmatic patients compared to control subjects. IL-33 expression correlates TNF-α e.g. a hallmark of inflammation. Airway epithelium, smooth muscle cells and endothelium are all sources of IL-33. When exposed to inflammatory conditions, *in vitro* cultured bronchial smooth muscle and epithelial cells increased their IL-33 expression, which surprisingly remained intracellular. Finally, glucocorticoid did not significantly reduce TNF-α-induced IL-33 expression.

Conclusions

Our study first describes IL-33 expression in asthma; it is increased in the lungs from asthmatics, and is enhanced under asthma-like *in vitro* conditions. IL-33 originates from structural cells of the airways and its expression does not respond to classic anti-inflammatory drug, thus reinforcing its relevance as a potential therapeutic target to treat asthma.

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